Joints of the Lower Extremity

Introduction

This lesson focuses on the injuries and joint pathologies of the hip, knee, and ankle. The emphasis of the hip is on femur factures and the vascular supply. The lesson's main bulk begins with the anatomy and physiology of the knee, then turns its attention to injuries of the knee and the physical exam maneuvers behind them. Rounding out the lesson are the relevant anatomy of the ankle, ankle sprains, and common diseases that receive honorable mention, but that aren't high yield (and that are absent from the video).

Hip 1: The Hip Joint

The hip is a **ball-and-socket** joint formed by the **femoral head** (the ball) and the **acetabulum** (the socket) of the pelvis. Being a ball-and-socket joint, the hip, like the shoulder, is able to move in many planes. The shoulder is more agile than the hip, and is out to the side of the axial skeleton, while the hip is positioned under the body to support the weight of the axial skeleton and to balance the torso during locomotion. Hip joint pathologies are mainly about fractures and the vascular supply to the femoral head. We go over fractures and avascular necrosis in detail, then have some lower-yield hip diseases that are generally more relevant for study in the clinical sciences.

Hip 1: Hip Fractures

Hip fractures generally occur near the joint, at the femoral neck or trochanters. In a hip fracture, the affected leg is **shortened** and **externally rotated**. Memorize this fact. Don't attempt to justify why or how it happens. You will constantly be assessed through your career regarding hip dislocation and hip fracture findings. Fracture is shortened and externally rotated. Dislocation is the "other one," so therefore shortened and internally rotated. Fractures rotate out.

The joint capsule is the space that consists of the head of the femur (the ball) and the acetabulum of the pelvis (the socket). The joint capsule is irrigated by a branch of the femoral artery called the **medial femoral circumflex artery**, which supplies the neck and head of the femur. This branch wraps around the bone and projects small arteries proximally from a distal arterial source. That means that the thing farthest from the blood supply, and the most likely to suffer ischemic injury, is the joint space. That also means that if there is a fracture of the femur between the trochanter and the joint itself, the breaking of bone could sever that artery.

Therefore, there are two types of hip fractures: extracapsular fractures that can't compromise the hip joint's blood supply, and intracapsular fractures that can.

Intracapsular fractures occur within the joint space, within the joint capsule, and involve the neck and head of the femur, which articulates with the acetabulum. Because of the tenuous blood supply coming from the **medial femoral circumflex artery** (the proximal femur head is supplied from an artery that originates distally, so that a fracture could result in total loss of blood supply), an intracapsular fracture can result in **avascular necrosis**. Other problems of healing (healing requires a blood supply) include **nonunion** and **malunion** also caused by compromised vascular supply.

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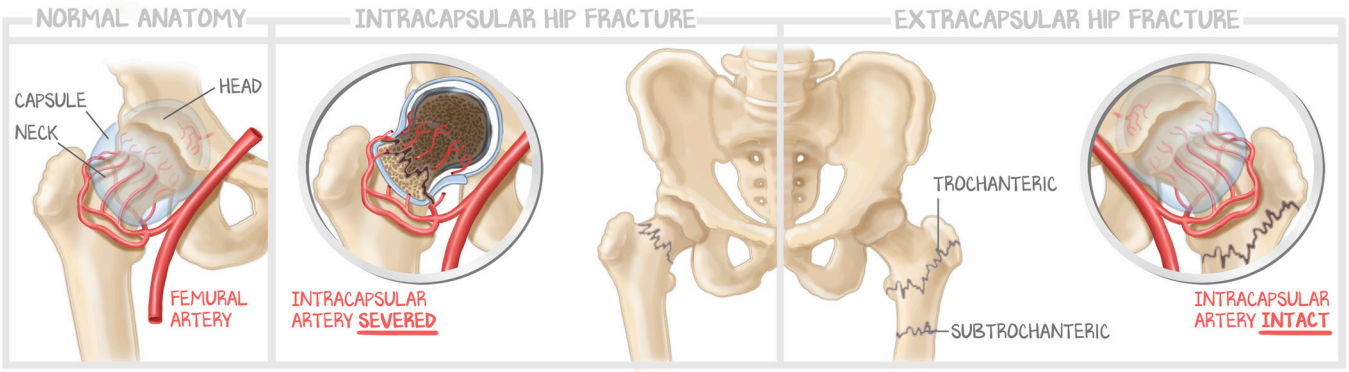

Figure 5.1: Hip Fractures
The different nomenclature of hip fractures—intracapsular vs. extracapsular—and the further delineation of extracapsular fractures—trochanteric and subtrochanteric. An intracapsular fracture is at risk of avascular necrosis but an extracapsular fracture is not.

Extracapsular fractures are those that occur outside of the joint space. If the fracture occurs between the greater and lesser trochanter, it is called an **intertrochanteric fracture** (this is synonymous with trochanteric facture). If the fracture occurs in the shaft of the femur, it is called a **subtrochanteric fracture**. These fractures have less risk of avascular necrosis, nonunion, and malunion because there isn't a tenuous blood supply.

Hip 2: Avascular Necrosis of the Hip

Avascular necrosis (also called osteonecrosis) happens as a result of compromise of bone arterial irrigation, resulting in the death of bone and marrow cells. The vascular supply to the head of the femur is the most tenuous, and is where this process tends to occur. We cited avascular necrosis of the hip as a complication of trochanteric fractures, but there are other medical (i.e., nontraumatic) causes of avascular necrosis, namely sickle cell disease, lupus, and corticosteroid use. The mechanism behind avascular necrosis induced by **corticosteroid** use and **lupus** are not well understood. **Sickle cell disease** causes sickling of red blood cells, which in turn causes vaso-occlusion of capillaries. This compromised blood flow puts tissue (hips, scaphoid) at risk for avascular necrosis. In avascular necrosis, the hip dies. Dying tissue hurts (**groin pain**). A dying or dead hip also doesn't do what it is supposed to do very well, so there will be a **decreased range of motion** (inability to internally rotate or abduct, which is very different than the fracture), **inability to bear weight**, or **pain with activity**. Surgery is the only option for correction.

Hip #3: Trochanteric Bursitis

This is a low-yield disease for testing, but high-yield exam for life. It is the **most common cause** of **lateral** hip pain in adults. The gluteus medius and minimus insert on the greater trochanter. There is a trochanteric bursa near to where they insert. Both **tendinitis** of these gluteus muscles and **inflammation of the bursa** can lead to the same presentation. There is **focal tenderness** over the greater trochanter. Having the patient cross the unaffected leg over the straightened affected leg, then bending at the waist can increase the pain, nailing the diagnosis. Rest and NSAIDs are the treatment.

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Hip #4: Pediatric Hips

These conditions are more commonly tested in the clinical sciences, needing to be recognized and diagnosed. There are too few mechanisms involved to make them targets for licensure in the basic sciences. Like trochanteric bursitis, they are included for thoroughness.

DISEASE	AGE	PATIENT	DIAGNOSIS	TREATMENT
Developmental dysplasia of the hip	Newborn	Clicky hip	U/S	Harness
Legg-Calvé-Perthes	6	Insidious antalgic gait	X-ray	Cast
Slipped capital femoral epiphysis	14	Growth spurt Nontraumatic pain	Frog-eg X-ray	Surgery

Table 5.1:

Knee #1: The Normal Knee

Bones. The knee joint is formed by four bones: the **femur** from the thigh, the **tibia** and **fibula** from the foreleg, and the **patella**, the free-floating bone known as the knee cap, which sits out in front of the knee joint. The bones are connected by tendinous insertions of muscles and by ligaments.

Muscles. The knee is supposed to do only one plane of motion—flexion and extension. The knee is flexed (bent) when the pull-muscles on the back of the thigh (the hamstrings) contract, and the knee is extended (straightened) when the push-muscles on the top of the thigh (the quadriceps) contract. The quadriceps tendon travels over the patella and inserts on the tibia. Above the patella, that same tendon is known as the quadriceps tendon, and as the **patellar tendon** below the patella. The patellar tendon inserts onto the **tibial tuberosity**. The muscles from the thigh traverse the knee joint and insert on the tibia of the foreleg.

Ligaments. The knee is prevented from doing other planes of motion outside flexion and extension by **ligaments**. These ligaments stabilize the knee joint, preventing the tibia from moving anteriorly, posteriorly, laterally, medially, or twisting.

Collateral ligaments. The ligaments that resist side-to-side motion are named based on their orientation to the femur. The **lateral epicondyle** of the femur is where the **lateral collateral ligament** (LCL) originates; the LCL attaches to the lateral bone of the foreleg, the fibula. The LCL resists the lateral motion of the tibia/fibula. The **medial epicondyle** is where the **medial collateral ligament** (MCL) originates; the MCL attaches to the medial bone of the foreleg, the tibia. The MCL resists the medial motion of the tibia/fibula. These are extracapsular ligaments.

Cruciate ligaments. The ligaments that resist "forward and back" motion of the tibia are named for where they originate on the **tibia**. The **anterior cruciate ligament** (ACL) attaches from the **anterior of the tibia** to the posterior, medial condyle of the femur. The ACL prevents excessive anterior motion of the tibia anteriorly. The **posterior cruciate ligament** (PCL) attaches from the **posterior of the tibia** to the anterior lateral condyle of the femur. The PCL prevents excessive posterior motion of the tibia posteriorly. These are intracapsular ligaments.

Menisci. The knee is also cushioned by **menisci**, two crescent-shaped pads of cartilage that rests between the tibia and femoral condyles. It is squishy cartilage that prevents the femur and tibia bones from rubbing against each other. There are two menisci in each knee, a medial and a lateral, which correspond to the medial and lateral condyles.

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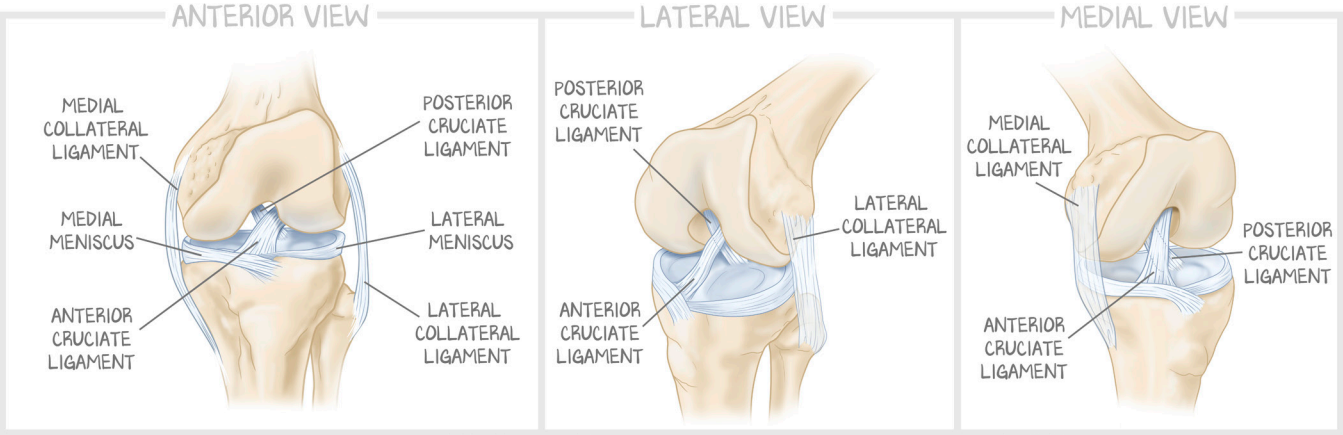

Figure 5.2: The Knee Joint
Using anterior, lateral oblique, and medial oblique views, you can identify each of the ligaments and menisci. Ligaments are named for their location on the tibia. The anterior cruciate ligament inserts on the anterior of the tibia and prevents anterior movement of the tibia relative to the femur. The posterior cruciate ligament is posterior on the tibia and prevents posterior movement of the tibia relative to the femur.

Knee 2: Ligament Injuries

Knee injuries are primarily damage to the stabilizers of the knee joint, damage to the ligaments. Ligaments tear when they are overstretched. They overstretch when they are forced to do the thing they are supposed to prevent, with a force that exceeds their capacity to resist. Ligaments, unlike muscles, don't get stronger with use. Ligaments get stretched, then snap back to their original position. If they get overstretched, if the force applied exceeds their ability to resist, they break. For example, if the tibia is moved too far anterior, with the force coming from the posterior aspect, the anterior cruciate ligament is stretched. What should happen is that it does not break, and the tibia movement is restricted, and the knee stays normal. If enough force is applied, the tibia moves too far anterior, and the ACL tears. The force that tears a ligament comes from the side of the knee that is opposite the side of the ligament's actions. A force from the posterior aspect of the knee stretches and tears the anterior cruciate ligament. For some, this is really easy to visualize. For most, even though it is relatively simple, doing this visualization in your head is a major pitfall—it's really easy to get it backwards.

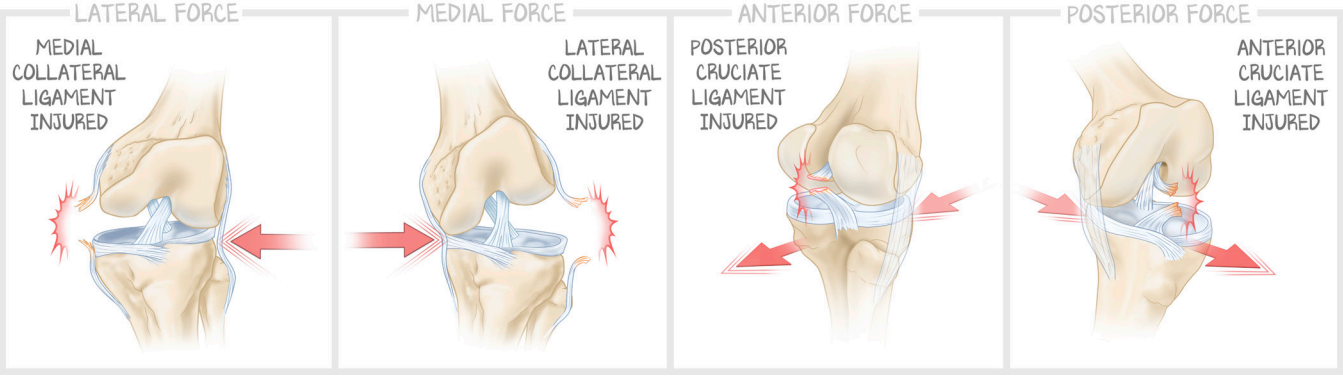

Figure 5.3: Force Injures the Ligament Opposite the Site of Impact
If stretched too far, ligaments will tear. Ligaments restrict the movement of the tibia relative to the femur. Ligaments get stretched when an external force is applied to the knee. A force originating behind the knee will collide with the posterior of the knee. A force originating behind the knee must travel anteriorly to collide with the posterior of the knee. That force drives the tibia forward. The anterior cruciate ligament restricts the anterior movement of the tibia and so is torn by the force delivered to the posterior of the knee.

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Damage to the **medial collateral ligament** is caused by a force applied to the **lateral** aspect of the knee, pushing the femur and the tibia medially, opening the knee joint on the medial side. The ligament breaks if enough force is applied. When it breaks, the medial collateral ligament no longer resists the medial movement of the bones. When gentle force is applied to knee joint under examination, there will be a laxity (a loss of stability) opposite to the pressure applied. If applied to the medial aspect, there is no laxity (the lateral collateral ligament is intact). If applied to the lateral aspect (the same side as the original trauma that caused the damage) there will be laxity on the medial side. The application of force to the lateral aspect is called a valgus stress. Memorize vaLgus Lateral, or "vaLateralgus stress," and know that the varus stress is the other one. Applying a lateral force assesses the medial collateral ligament; a positive test is laxity of the joint or a widening of the medial femur from the fibula. MCL injuries are common in tackling sports and soccer—the lateral aspect of a players knee is the outer layer, exposed to collision.

Damage to the **lateral collateral ligament** is caused by a force applied to the **medial** aspect of the knee, pushing the femur and fibula laterally. The ligament tears if enough force is applied. When it tears, the lateral collateral ligament no longer resists the lateral movement of the bones. The force that causes the tear is delivered medially. The force that results in lateral laxity after a lateral ligament is torn is also from the medial aspect. The application of a medial force to assess the lateral ligament is called a **varus** force. A positive test is if there is laxity of the lateral ligament, a widening of the tibia and femur. Since players don't generally get between each other's legs, LCL tears are rare in isolation. They tend to occur with massive trauma that destroys multiple ligaments.

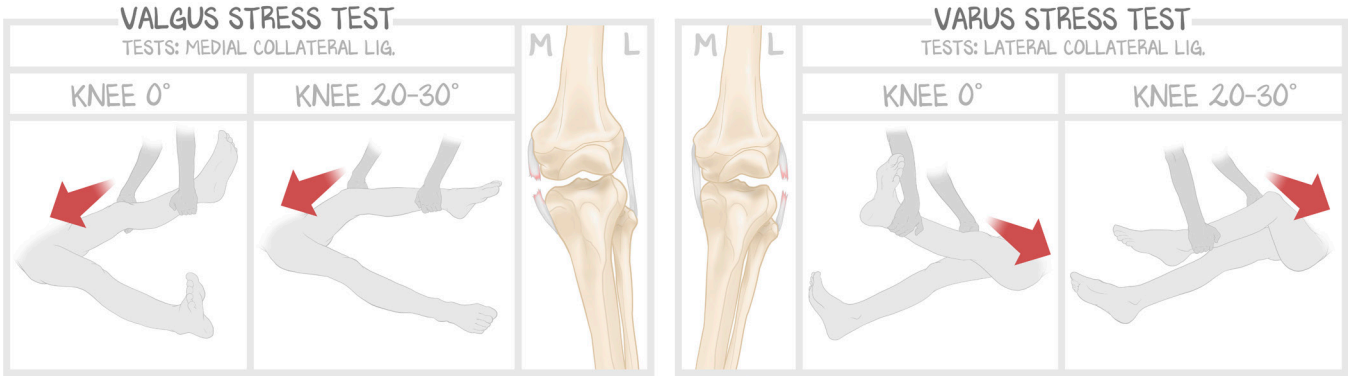

Figure 5.4: Valgus and Varus Stress Tests
The examiner will lift the patient's leg either extended or with the knee flexed at a 30° angle. A "vaLateralgus" stress is applied, then a varus stress. The knee is assessed for laxity. If there is a widening of the knee with a vaLateralgus stress, the median collateral ligament is defective. If there is knee widening with a varus stress, the lateral collateral ligament is defective.

Damage to the **anterior cruciate ligament** is caused by a **posterior force** to the knee, usually below the knee against the tibia. This drives the tibia forward. The ACL should hold it in place, but if enough force is applied, it tears. Once torn, there is no resistance to the anterior movement of the tibia. A noncontact injury can occur as well, as with a **sudden pivoting movement** at high speeds, which can yank on the ligament and shear it from a twisting axial force. There is often a **rapid onset effusion** and instability of the knee. The **anterior drawer test** (or Lachman) is designed to assess the integrity of the ACL. A positive test is the finding of laxity, or the ability to go farther on the affected side than on the unaffected. This force is a posterior force moving anteriorly. Practically, this force is applied with the examiner in front of the patient, exerting a pulling force on the tibia.

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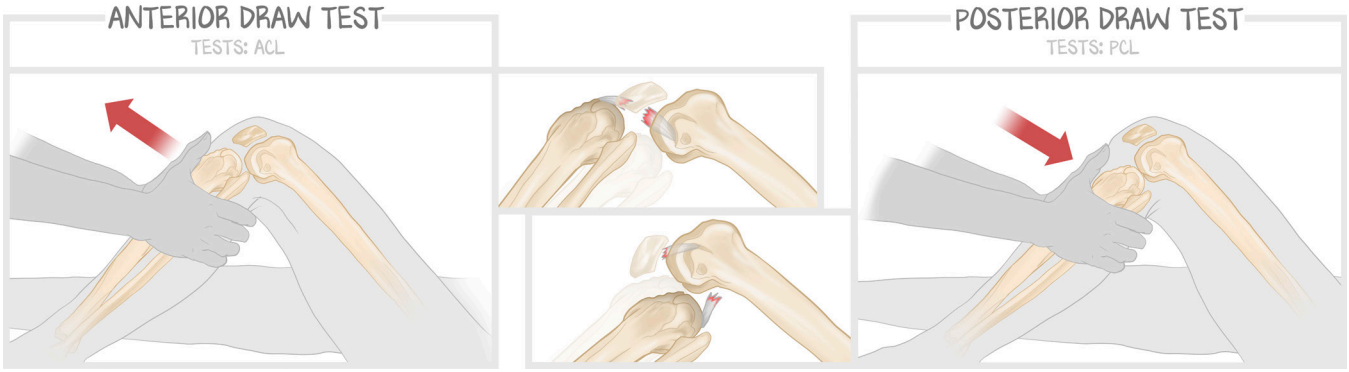

Figure 5.5: Drawer Testing
The Lachman is at 30°, the anterior and posterior drawer test at 90°. Since these maneuvers will be performed together, it makes sense to use the 90° position. Naming the test will not be the target of test questions. Recognizing that pulling the tibia forward assesses the ACL and pushing the tibia assesses the PCL is all that is necessary.

Damage to the **posterior cruciate ligament** is caused by an **anterior force** to the knee, usually below the knee against the tibia. This is often seen in **car accidents** when the knee strikes the dashboard. This drives the tibia backwards. The PCL should hold it in place, but if enough force is applied, it tears. Once torn, there is no resistance to the posterior movement of the tibia. The **posterior drawer test** is designed to assess the integrity of the PCL. A positive test is the finding of laxity, or the ability to go farther on the affected side than on the unaffected side. This force is an anterior force moving posteriorly. Practically, this force is applied with the examiner in front of the patient, pushing on the tibia.

Knee #3: Meniscal Injuries

The menisci are padding, shock absorbers, that help prevent the internal rotation and external rotation of the knee. Damage to these structures occurs by **twisting the knee while the foot is planted**. That sounds a lot like pivoting (ACL tear mechanism). However, unlike the ACL tear, the effusion develops slowly; the patient can still walk, but it just **hurts to squat**, **twist**, or **pivot**. The patient will describe a **locking** or a **catching** sensation. If the motion were an **external rotation** it would result in a **medial tear**. If the motion were an **internal rotation** there would be a **lateral tear**. McMurray and Apley tests will be positive, where the mechanism of injury is repeated, looking for a popping or exacerbation of pain.

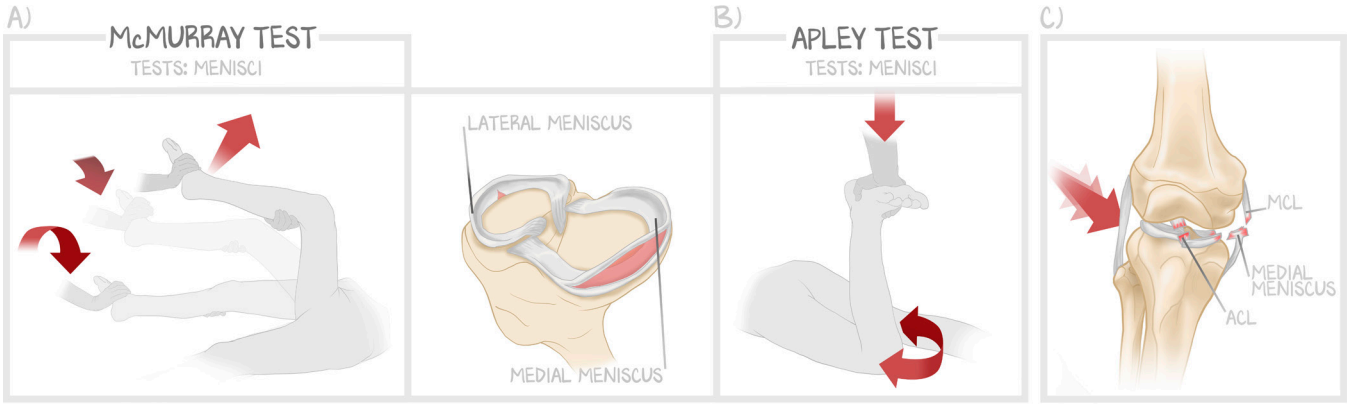

Figure 5.6: Meniscus Injuries
(a) The McMurray test is performed by starting with the knee flexed. The examiner applies a valgus stress while extending the knee at the same time the foot is externally rotated. (b) Apley's compression test is a downward pressure on flexed knee in the prone position and then the leg is internally and externally rotated. (c) The unhappy triad demonstrating that injuries may not necessarily occur in one plane, and how a severe force can tear multiple structures at once.

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A specific injury, the "unhappy triad," occurs in sports injury where a posterolateral force is applied to a planted leg. This happens in soccer, baseball slides, and football tackles. The lateral force strains the medial collateral ligament, which tears. The posterior force strains the anterior cruciate ligament. And the stretch caused by the opening of the femur-tibia bones on the medial meniscus causes it to tear. **MCL + ACL + medial meniscus = Unhappy Triad.**

Knee #4: Inflammation

These knee issues are more appropriate for Clinical Sciences. They have appeared on Basic Science examinations, so are included here. They are not present in the video because they take too much time to say, and are too low yield for Basic Science.

Prepatellar bursitis. Prepatellar bursitis is an inflammation of the bursa on top of the patella. This occurs as a **repetitive trauma** or **excessive pressure from kneeling** (also called "housemaid's knee"). The patient presents with a **tenderness and swelling** that is **anterior to the patella** or patellar tendon.

Baker's cyst. A baker's cyst is an outpouching of the semimembranosus bursa in the popliteal fossa. It will be palpated in the popliteal fossa. It is otherwise asymptomatic until it ruptures. If it ruptures it can present like a DVT, or it can result in hemarthrosis.

Patellofemoral pain syndrome. The patellar tendon is contiguous with the quadriceps tendon. This tendon originates from the quadriceps in the thigh, rides over the patella, and inserts onto the tibial tuberosity. This is how contraction of the quadriceps causes knee extension. Patellofemoral pain syndrome is an overuse injury (it is also called "runner's knee") that causes pain at the insertion point of the patellar tendon (the tibial tuberosity) with any repetitive motion that bends the knee.

Osgood-Schlatter's disease. Osgood-Schlatter's disease is also called tibial tuberosity avulsion. When children perform repetitive activity using the knee (running, jumping) the quadriceps tug on the tibial tuberosity. Because they are children, there is an active growth plate. Repeated tension on the growth plate can cause it to tear, or avulse. The patient will present with a painful knee and a firm bump (of bone) located at the tibial tuberosity.

Iliotibial band syndrome. The IT band runs longitudinally along the lateral aspect of the thigh from the iliac crest to the proximal tibia on the lateral side. It is also an overuse injury commonly seen in **runners**. It is caused by friction of the IT band against the lateral femoral condyle. Recognizing overuse injury + lateral knee pain, then knowing it is the lateral condyle, is enough.

The Ankle and Foot

There are many ligaments at the ankle. Some attach to the fibula and tibia—the tibiofibular ligaments. There is one in front (anterior) and one in back (posterior). Some attach the fibula to the talus bone (the talofibular ligaments). There is one in front (anterior) and one in back (posterior). The anterior ligaments**,** the **anterior tibiofibular ligament** and the **anterior talofibular ligament**, are the ones that will come up in ankle sprains. There are other ligaments and there are other bones. Do not learn any others, except for the ligament that connects the fibula to the calcaneus bone (the **calcaneofibular ligament**).

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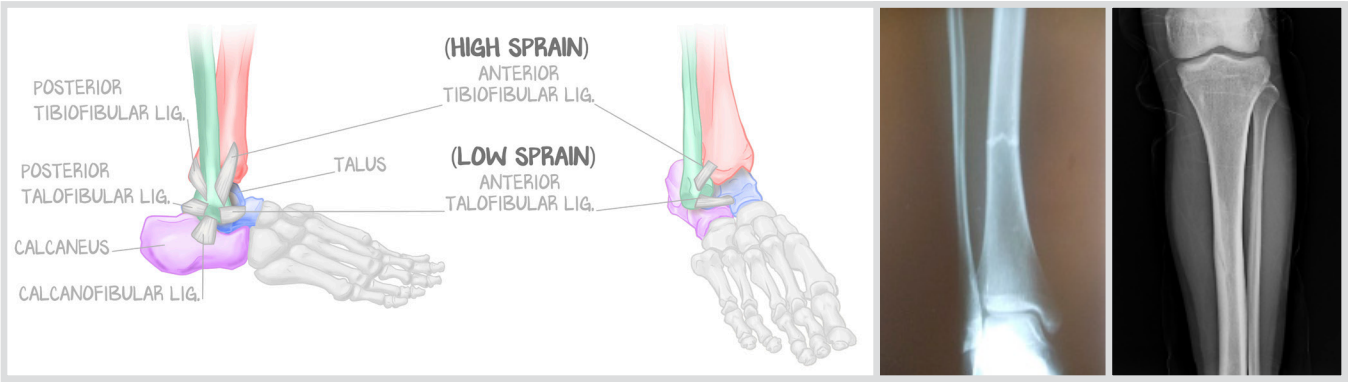

Figure 5.7: The Ankle and Shin
(a) The bones you should recognize (talus, calcaneus), as well as the ligaments you should be aware of, knowing that there are five (i.e., ripe for a test question) but only two really matter. If stuck, choose between the two anterior ligaments. (b) An X-ray of a stress fracture of the tibia. (c) An X-ray of the tibia in shin splints (normal).

Tendons get strained, ligaments get sprained. A **low-ankle sprain** is the **most common** ankle sprain. It is caused by inversion of a plantar-flexed foot. This strains the **anterior talofibular ligament** (from the fibula to the talus). A **high-ankle sprain** is a sprain of the anterior inferior tibiofibular ligament (tibia to fibula), caused by forced dorsiflexion and eversion of the ankle.

Stress fracture vs. shin splints. Both of these injuries have similar presentations and are caused by a similar stressor. The underlying pathology, however, is quite different. Both will be caused by an abrupt increase in physical activity that uses the muscles of the foreleg—think a runner or military marches. They both present with shin pain. A **stress fracture** is a fracture of the tibia or fibula. The increased pounding on these bones causes the fracture, but only in weakened bones—**inadequate Ca/Vit D** or a **female athlete triad** (low calories, amenorrhea, low bone density). There is an insidious onset of localized pain, with **point tenderness** over the fracture site. Shin splints, or **medial tibial stress syndrome** is also caused by repetitive activity, but without bone density risk factors. This is NOT caused by a fracture, but rather bone resorption outpaces bone formation in the tibial cortex. It hurts, but is not broken. The pain is **vague** and **diffuse**.

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